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Cellular Track Model of Biological Damage to Mammalian Cell Cultures From Galactic Cosmic Rays

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Introduction

The quality factor (QF) as defined in International Commission on Radiological Protection report no. 26 (ICRP 26, ref. 1) or in International Commission on Radiation Units and Measurements report no. 40 (ICRU 40, ref. 2) is not expected to be a valid method for assessing the biological risk for deep space missions where the high-energy heavy ion (HZE) particles of the galactic cosmic rays (GCR) are of major concern. No human data for cancer induction from the HZE particles exist, and information on biological effectiveness is expected to be taken from experiments with animals and cultured cells (ref. 3). Experiments with cultured cells (refs. 4–6) indicate that the relative biological effectiveness (RBE) of the HZE particles is dependent on particle type, energy, and the level of fluence. Use of a single parameter, such as linear energy transfer (LET) or lineal energy (see ref. 2), to determine radiation quality will therefore represent an extreme oversimplification for GCR risk assessment.

Katz has presented a theoretical model (refs. 7 and 8) that predicts the correct RBE behavior as observed in recent experimental studies using track-segment irradiations with heavy ions on cultured mammalian cells. Cells at risk in deep space will be subject to a complicated mixture of particles varying in composition with the amount and type of shielding surrounding them. The fluence levels in space are such that a single cell will likely be exposed to only one ion encounter over an extended period. Katz has developed the ion-kill mode of cell death or transformation that corresponds to low-fluence exposures. The delta-ray (energetic electrons produced in ion collisions) radial dose distribution surrounding the ion path is assumed to initiate the biological damage, and the cell response to the radiation field is parameterized using target theory and results from gamma-ray and track-segment irradiations. The level of damage for a mixed-radiation field is determined by the cellular response parameters and the local flux of particles. The Langley Research Center has developed a deterministic transport code for calculating the differential flux of ions behind natural and protective radiation shielding exposed to the GCR spectrum (refs. 9–13). In this paper we consider the biological damage to mammalian cell cultures expected for 1 year in deep space at solar minimum behind various depths of aluminum shielding using the Katz cellular damage model and the Langley GCR code. Cell death and neoplastic transformations for C3H10T1/2 cells (mouse embryo cells) are considered for typical levels of spacecraft shielding. The results of this study must be considered preliminary in that the transport code is in an early stage of development (ref. 13).

Galactic Cosmic Ray Transport

The GCR spectrum consists of energetic ions with charges Z from 1 to 90. In passing through spacecraft shielding and tissue these ions will lose energy through atomic interactions, and occasionally through nuclear fragmentation events of GCR ions (projectile fragmentation) and atomic nuclei in the shielding (target fragmentation). The transport equation for the projectile ions and their secondaries is written in the straight-ahead approximation as (ref. 10)

$$\left[\frac{\partial}{\partial x} - \frac{\partial}{\partial E}\tilde{S}_j(E) + \Sigma_j(E)\right]\Phi_j(x,E) = \sum_{k>j}\Sigma_{jk}(E)\ \Phi_k(x,E) \tag{1}$$

where $\Phi_j(x,E)$ is the flux of ions of type j with atomic mass A_j and charge Z_j at x moving along the x-axis with energy E (given in units of MeV/amu), $\tilde{S}_j$ is the change in E per unit distance, Σ_j is the macroscopic nuclear absorption cross section, and Σ_{jk} is the macroscopic fragmentation cross section for the production of ion j from ion k.

Equation (1) has been solved by Wilson (refs. 9 and 10) using a characteristic transformation and perturbative methods. The perturbative solution represents the production of secondary and later generation ions as the GCR spectrum is propagated through the shield. The major shortcoming in the GCR code is the limited data base for the fragmentation parameters. This includes neglect of mass 2 and 3 projectile fragments. The energy dependence of the fragmentation parameters is ignored. Improvements in the treatment of neutron secondaries are also needed, and meson production is not included. Details on the method of solution and the physical inputs are found in references 9–13.

The target-fragmentation fields are found in closed form in terms of the collision density (ref. 9) since these ions are of relatively low energy. Away from any interfaces, the target fields are in a local equilibrium and may be written as

$$\Phi_\alpha(x,E_\alpha:E_j) = \frac{1}{S_\alpha(E_\alpha)}\int_{E_\alpha}^{\infty}\frac{d\Sigma_{\alpha j}(E',E_j)}{dE'} \times \Phi_j(x,E_j)\ dE' \tag{2}$$

where the subscript α denotes the target-fragment

type, S_α denotes the stopping power, and E_α and E_j are given in units of MeV.

The particle fields of the projectiles and the projectile and target fragments determine the level and type of radiological damage at the end point of interest. The relationship between the fields and the cellular response is considered next within the Katz cellular track model.

Cellular Track Model

The cellular track model of Katz has been described extensively (refs. 7, 14, and 15). Here we outline its basic concepts and consider the extension to the mixed-radiation fields seen in space. The biological damage from passing ions is caused by delta-ray production. Cell damage is separated into a grain-count regime, where damage occurs randomly along the ion path, and a track-width regime, where the damage is distributed like a "hairy rope." The response of the cells is described by four cellular parameters, two of which (m, the number of targets per cell, and D_0, the characteristic X-ray dose) are extracted from the response of the cellular system to X-ray or gamma-ray irradiation. The other two (σ_0, interpreted as the cross-sectional area of the cell nucleus within which the damage sites are located, and κ, a measure of the size of the damage site) are found from survival measurements with track-segment irradiations by energetic charged particles. The transition from the grain-count regime to the track-width regime takes place at $Z^{*2}/\kappa\beta^2$ on the order of 4, where Z^* is the effective charge number and β is the velocity. The grain-count regime is at the lower values of $Z^{*2}/\kappa\beta^2$ and the track-width regime is at the higher values.

To accommodate for the capacity of cells to accumulate sublethal damage, two modes of inactivation are identified: ion-kill (intratrack) and gamma-kill (intertrack). For cells damaged by the passage of a single ion, the ion-kill mode occurs. The fraction of cells damaged in the ion-kill mode is taken as $P = \sigma/\sigma_0$, where σ is the single-particle-inactivation cross section and P is the probability of the damage in the ion-kill mode. Cells not damaged in the ion-kill mode can be sublethally damaged by the delta rays from the passing ion and then inactivated in the gamma-kill mode by cumulative addition of sublethal damage due to delta rays from other passing ions. The surviving fraction of a cellular population N_0, whose response parameters are m, D_0, σ_0, and κ, after irradiation by a fluence of particles F is written as (ref. 7)

$$\frac{N}{N_0} = \pi_i \times \pi_\gamma \tag{3}$$

where the ion-kill survivability is

$$\pi_i = e^{-\sigma F} \tag{4}$$

and the gamma-kill survivability is

$$\pi_\gamma = 1 - \left(1 - e^{-D_\gamma/D_0}\right)^m \tag{5}$$

The gamma-kill dose fraction is

$$D_\gamma = (1 - P)D \tag{6}$$

where D is the absorbed dose. The single-particle-inactivation cross section is given by

$$\sigma = \sigma_0 \left(1 - e^{-Z^{*2}/\kappa\beta^2}\right)^m \tag{7}$$

where the effective charge number is

$$Z^* = Z\left(1 - e^{-125\beta/Z^{2/3}}\right) \tag{8}$$

In the track-width regime, where $P > 0.98$, we take $P = 1$.

For cell transformation the fraction of transformed cells per surviving cell is

$$T = 1 - \frac{N'}{N_0'} \tag{9}$$

where N'/N_0' is the fraction of nontransformed cells, and a set of cellular response parameters for transformations m', D_0', σ_0', and κ' are used. The RBE at a given survival level is given by

$$RBE = \frac{D_X}{D} \tag{10}$$

where

$$D_X = -D_0 \ln\left[1 - \left(1 - \frac{N}{N_0}\right)^{1/m}\right] \tag{11}$$

is the X-ray dose at which this level is obtained. Equations (3)–(11) represent the cellular track model for monoenergetic particles. Mixed-radiation fields have been considered previously in the Katz model. (See, for example, ref. 14.) Next we consider placing the model in terms of the particle fields described above.

Cell Damage for the GCR Spectrum

In order to apply the cellular track model to the mixed-radiation fields seen in space, we need to make the appropriate replacement of the cross section and

particle fluence number (σF) with the particle field quantities and their corresponding cross sections. The ion-kill term, which will now contain a projectile source term (including projectile fragments) and a target fragment term, is written as

$$\sigma F = \sum_j \int dE_j \; \Phi_j(x, E_j) \; \sigma_j(E_j)$$

$$+ \sum_\alpha \sum_j \int dE_\alpha \; dE_j \; \Phi_\alpha(x, E_\alpha : E_j) \; \sigma_\alpha(E_\alpha) \quad (12)$$

where the second term is the contribution of nuclear fragments produced locally in the biological medium. This may be written in terms of an effective-action cross section σ^* for the passing ion, whose track is dressed by the local target fragments (nuclear stars), as

$$\sigma F = \sum_j \int dE_j \; \Phi_j(x, E_j) \; \sigma^*(E_j) \quad (13)$$

The gamma-kill dose fraction becomes

$$D_\gamma = \sum_j \int dE_j \; \Phi_j(x, E_j) \; [1 - P_j(E_j)] \; S_j(E_j)$$

$$+ \sum_j \sum_\alpha \int dE_j \; dE_\alpha \; \Phi_\alpha(x, E_\alpha : E_j)$$

$$\times \; [1 - P_\alpha(E_\alpha)] \; S_\alpha(E_\alpha) \quad (14)$$

Equations (12) and (14) are used in equations (4) and (5), respectively. The summations over all particle types in equations (12) and (14) represent the addition of probabilities from all ions in the radiation field that contribute to the end point under study.

Results and Discussion

Cellular parameters obtained in reference 8 for survival and neoplastic transformations of C3H10T1/2 cells obtained from the experiments of Yang et al. in reference 4 are given in table 1. We note that the large uncertainties in the transformation data of Yang should lead to a similiar uncertainty in the transformation parameters. Parameter sets were found from data for instantaneous and delayed plating of the cells after the irradiation. Here only the delayed plating case is considered. General agreement with the measured RBE values was found using these parameter sets (ref. 8). The single-particle-inactivation cross section neglecting the target fragmentation of equation (12) is shown in figures 1 and 2 for cell death and cell transformation, respectively, as a function of the energy of the passing ion. The target-fragmentation contribution (the second term in eq. (12)) for protons has been evaluated as shown in figures 3 and 4. For protons the effects of the target fragments (dashed line, second term in eq. (12)) dominate over the proton direct ionization (dotted line) at high energy. For high LET particles (low energy), the direct ionization dominates and target-fragmentation effects become negligible. A simple scaling by $A_j^{1/2}$ relates the proton target-fragment term to ions of mass A_j. The resulting effective-action cross sections for cell death and cell transformation are plotted in figures 5 and 6, respectively. We note that the low-energy ^{56}Fe component of the GCR spectra extends into the track-width regime where $\sigma > \sigma_0$ and is not represented in the present calculation. The error introduced by the present calculation is small.

The cellular track model was applied to predict the fraction of C3H10T1/2 cells killed or transformed for 1 year in deep space at solar minimum for typical spacecraft shielding. The GCR environment was taken from the Naval Research Laboratory code (ref. 16). Aluminum shielding was considered with a local region of tissue for the cell cultures. Tables 2 and 3 contain individual particle fluences and absorbed doses, respectively, for the protons, alpha

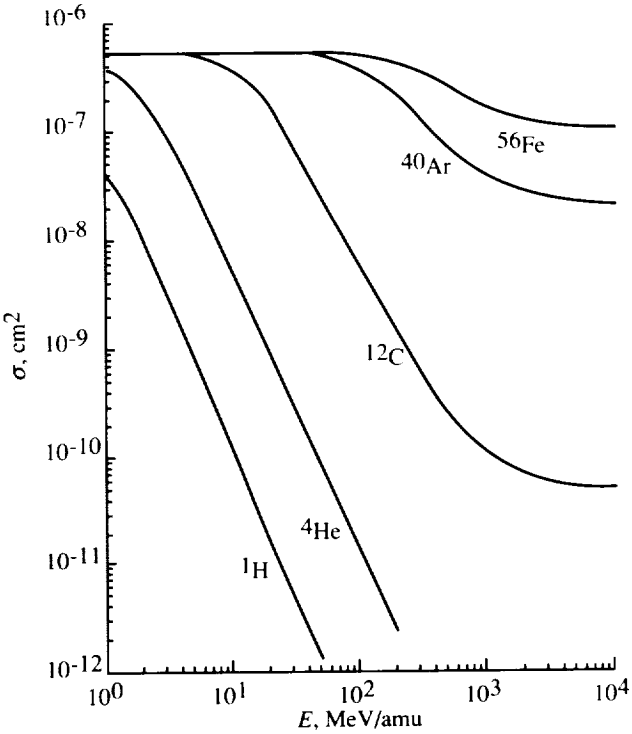

Figure 1. Cell-death cross sections for various ions in C3H10T1/2 cells according to the Katz model for direct ionization effects only.

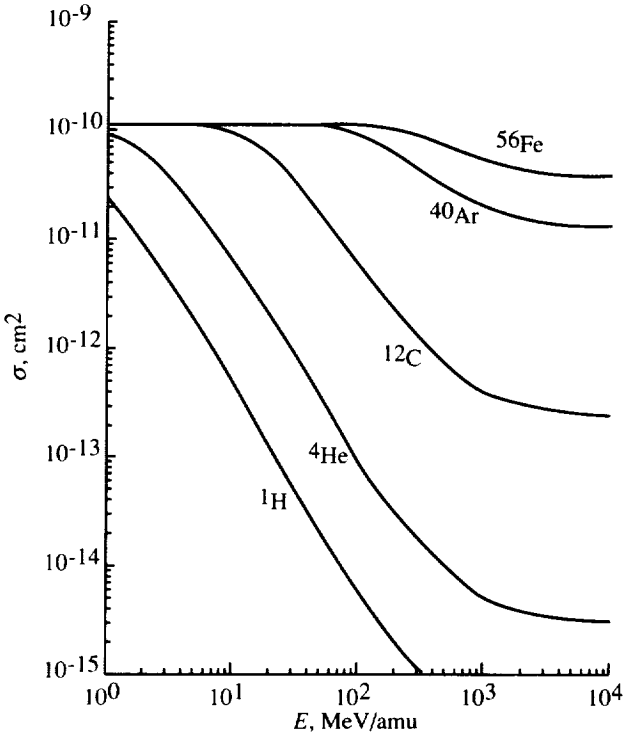

Figure 2. Cell-transformation cross sections for various ions in C3H10T1/2 cells according to the Katz model for direct ionization effects only.

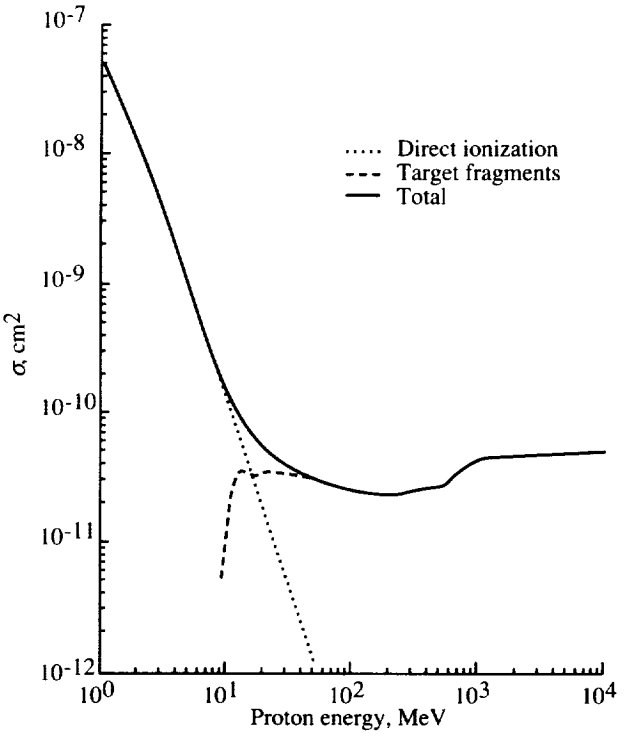

Figure 3. Cell-death cross sections including effects of nuclear reactions for protons in C3H10T1/2 cells according to the Katz model.

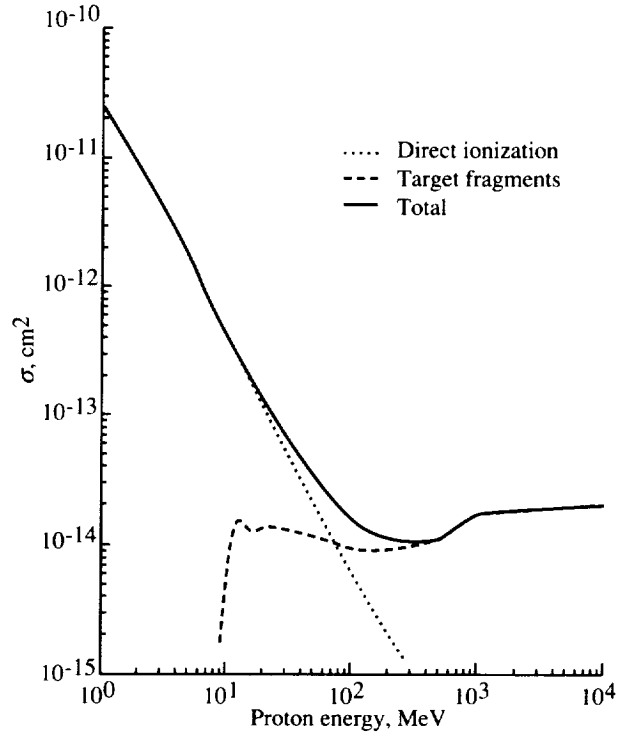

Figure 4. Cell-transformation cross sections including effects of nuclear reactions for protons in C3H10T1/2 cells according to the Katz model.

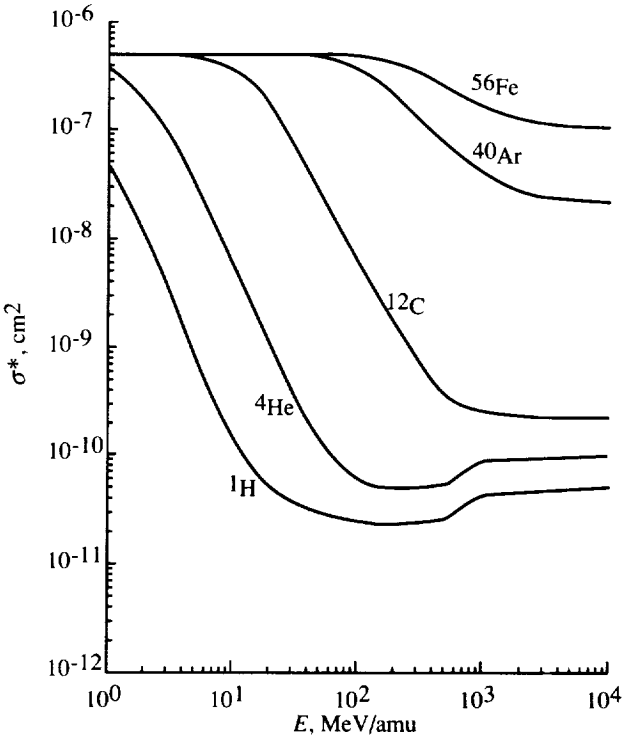

Figure 5. Effective cell-death cross sections including effects of nuclear reactions for various ions in C3H10T1/2 cells.

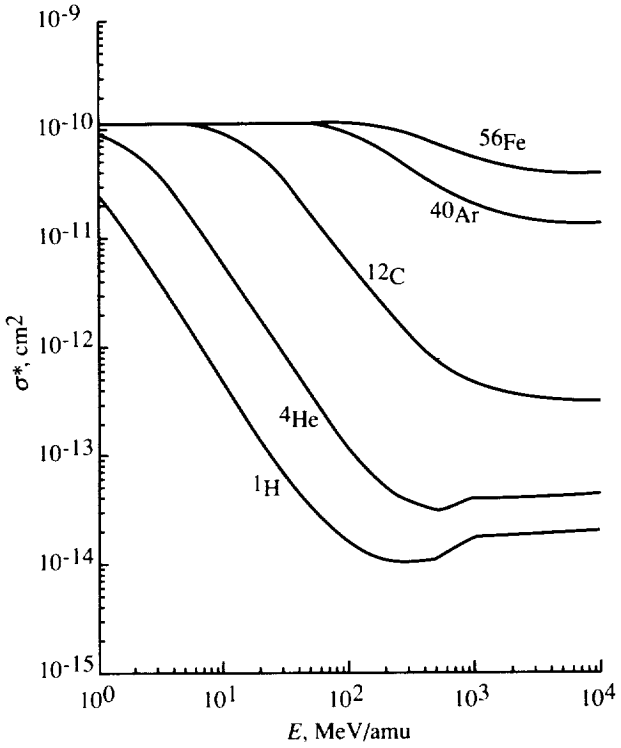

Figure 6. Effective cell-transformation cross sections including effects of nuclear reactions for various ions in C3H10T1/2 cells.

particles, $Z = 3$ to 9 ions (labeled L-Z) and $Z = 10$ to 28 ions (labeled H-Z) as determined by the Langley GCR code. Results for the fraction of C3H10T1/2 cells killed and transformed for 1 year at solar minimum are listed in tables 4 and 5, respectively. The gamma-kill mode was found to be of negligible importance in the calculations, indicating that biological damage in deep space from GCR particles at the cellular level will indeed result from the action of single particles. The importance of the target terms in biological effects for low LET protons and alpha particles is quite apparent. The results also indicate that the HZE component of the GCR spectrum is most damaging for small shielding depths. At large depths the HZE components break up and cause proton buildup with increasing shield depth. At large depths, the protons dominate the biological effects. In comparing individual charge components, we see that the H-Z particles have a reduced effectiveness for the transformation end point.

Also listed in tables 4 and 5 are the values of RBE versus depth for the two end points. In table 6 we present the present RBE values beside the average QF values taken from reference 13 using the same transport code. The fact that RBE and QF are nearly equal at small depths is somewhat coincidental. We note that the quality factor is independent of the fluence level, which is not true for the Katz model. The Katz model indicates a substantial increase in risk, at higher shielding levels, than the ICRP 26 quality factors (ref. 1).

The RBE values show a simple scaling with exposure time for the GCR particles as can be seen from equations (10), (11), and (4) when ion kill dominates. Here we find for

$$\frac{N}{N_0} \cong 1 \tag{15}$$

with

$$\sigma F << 1 \tag{16}$$

that

$$\mathrm{RBE} = \frac{D_0}{\mathrm{LET}} \sigma^{1/m} F^{[-1+(1/m)]} \tag{17}$$

Then, scaling the RBE as a function of duration in deep space to the 1-year value for a duration period of τ (with $F = n\tau$) gives

$$\mathrm{RBE}(\tau) = (\tau/\tau_1)^{[-1+(1/m)]} \mathrm{RBE}(\tau_1) \tag{18}$$

As a result, a one-hit ($m = 1$) system RBE becomes fluence independent as expressed by

$$\mathrm{RBE}(\tau) = \mathrm{RBE}(\tau_1) \tag{19}$$

a two-hit ($m = 2$) system is expressed by

$$\mathrm{RBE}(\tau) = \frac{\mathrm{RBE}(\tau_1)}{(\tau/\tau_1)^{1/2}} \tag{20}$$

and a three-hit ($m = 3$) system is expressed by

$$\mathrm{RBE}(\tau) = \frac{\mathrm{RBE}(\tau_1)}{(\tau/\tau_1)^{2/3}} \tag{21}$$

Results of this scaling approximation agree quite well with calculations using equations (3)-(15), as seen in table 7 where values obtained using the approximations of equation (18) are shown in parentheses as scaled from the 1-year RBE values taken from table 6, and results of the calculations are shown without parentheses. The extremely large RBE values that would be obtained for small values of τ are due to the choice of energetic photons as the reference radiation.

Concluding Remarks

A track structure model has been used with a deterministic galactic cosmic rays (GCR) transport code to predict the fractions of cell death and neoplastic transformations for C3H10T1/2 cells in deep space behind typical spacecraft shielding. Results indicate that the level of damage from the GCR particles does not attenuate appreciably for large amounts

of spacecraft shielding and that single particles acting in the ion-kill mode dominate the effects. The contribution from target fragments was seen to be important in assessing the biological effect of protons and alpha particles. The relative biological effectiveness (RBE) values obtained in this fluence-dependent model were found to be more severe than the ICRP 26 quality factors. A simple scaling law with the duration time in space was found to account for the change in RBE with fluence for the uniform GCR background.

The results of our calculations of the RBE for both cell death and cell transformation are remarkably close, especially when considering the very large difference in radiosensitivity parameters for these end points and the huge difference in the fraction of affected cells. About 1000 times as many cells are killed as are transformed. Nevertheless, 90 percent of the cells survive the conditions calculated here, and of these about 1 or 2 in 100 000 are transformed. Yet, this is not an insignificant fraction when we consider the number of cells per cubic centimeter in tissue and speculate about the number of cells transformed by radiation that are likely to lead to cancer.

The cell population in tissue, about 10^9 per cubic centimeter, suggests that after 1 year of exposure to GCR at solar minimum there would be about 10^4 transformed cells per cubic centimeter in tissue if *in vitro* and *in vivo* transformation parameters were equal. Additionally, we do not know the minimum number of transformed cells that can be injected into a mouse to induce a cancer. Clearly, priority must be assigned to the investigation of these questions. If one or two transformed cells were to lead to cancer, as in leukemia, we could not tolerate an exposure in which the transformation fraction exceeded 10^{-9}.

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Table 1. Cellular Response Parameters for C3H10T1/2 Cells

Cell-damage type	m	D_0, cGy	σ_0, cm^2	κ
Death	3	280	5.0×10^{-7}	750
Transformation	2	26 000	1.15×10^{-10}	750

Table 2. Flux for 1 Year at Solar Minimum Behind Aluminum Shielding

x, g/cm^2	Flux, particles/cm^2/yr, from—			
	Protons	Alphas	L-Z (*a*)	H-Z (*b*)
0	1.29×10^{-8}	1.24×10^{7}	1.09×10^{6}	3.0×10^{5}
1	1.31	1.21	1.05	2.8
2	1.33	1.18	1.01	2.7
3	1.34	1.15	.98	2.5
5	1.36	1.10	.91	2.2
10	1.40	.97	.77	1.7
20	1.43	.77	.57	1.1

[a] Z = 3 to 9 ions.
[b] Z = 10 to 28 ions.

Table 3. Dose for Solar Minimum Behind Aluminum Shielding

x, g/cm^2	Dose, cGy/yr, from—				
	Protons	Alphas	L-Z (*a*)	H-Z (*b*)	Total
0	6.2	3.0	2.8	5.0	17.1
1	6.3	2.7	2.5	3.6	15.1
2	6.8	2.6	2.4	3.3	15.1
3	7.1	2.6	2.3	3.1	15.0
5	7.6	2.4	2.1	2.7	14.8
10	8.5	2.1	1.7	2.0	14.3
20	9.5	1.7	1.1	1.1	13.4

[a] Z = 3 to 9 ions.
[b] Z = 10 to 28 ions.

Table 4. Fraction of C3H10T1/2 Cells Killed in Deep Space for 1 Year at Solar Minimum Behind Aluminum Shielding

x, g/cm^2	Fraction of cells killed of--					
	Protons	Alphas	L-Z (*a*)	H-Z (*b*)	Total	RBE
Including target fragments						
0	1.35×10^{-2}	0.46×10^{-2}	0.57×10^{-2}	2.08×10^{-2}	4.46×10^{-2}	7.1
1	.76	.15	.43	1.84	3.18	7.0
2	.80	.14	.41	1.69	3.04	6.9
3	.83	.14	.38	1.55	2.90	6.8
5	.88	.14	.34	1.32	2.68	6.7
10	.95	.12	.25	.91	2.22	6.5
20	1.02	.09	.15	.49	1.74	6.2
Without target fragments						
0	0.84×10^{-2}	0.37×10^{-2}	0.55×10^{-2}	2.08×10^{-2}	3.79×10^{-2}	6.7
1	.24	.06	.41	1.83	2.54	6.5
2	.28	.06	.39	1.68	2.41	6.3
3	.31	.06	.37	1.55	2.27	6.2
5	.35	.06	.33	1.31	2.04	6.1
10	.42	.05	.24	.91	1.61	5.7
20	.49	.04	.14	.48	1.15	5.3

[a] $Z = 3$ to 9 ions.
[b] $Z = 10$ to 28 ions.

Table 5. Fraction of C3H10T1/2 Cells Transformed in Deep Space for 1 Year at Solar Minimum Behind Aluminum Shielding

x, g/cm^2	Fraction of cells transformed					
	Protons	Alphas	L-Z (*a*)	H-Z (*b*)	Total	RBE
Including target fragments						
0	5.2×10^{-6}	2.0×10^{-6}	3.1×10^{-6}	7.5×10^{-6}	1.78×10^{-5}	6.4
1	3.5	1.0	2.7	6.7	1.39	6.4
2	3.7	1.0	2.6	6.2	1.35	6.3
3	3.9	.9	2.4	5.7	1.29	6.3
5	4.2	.9	2.2	4.9	1.22	6.2
10	4.7	.8	1.7	3.5	1.06	6.0
20	5.2	.6	1.1	2.0	.88	5.7
Without target fragments						
0	3.2×10^{-6}	1.6×10^{-6}	3.1×10^{-6}	7.5×10^{-6}	1.53×10^{-5}	6.0
1	1.4	.6	2.7	6.7	1.13	5.8
2	1.6	.6	2.5	6.2	1.09	5.7
3	1.8	.6	2.4	5.7	1.05	5.6
5	2.1	.5	2.1	4.9	.97	5.4
10	2.5	.5	1.6	3.5	.82	5.2
20	3.0	.4	1.0	2.0	.64	4.9

[a] $Z = 3$ to 9 ions.
[b] $Z = 10$ to 28 ions.

Table 6. Comparison of ICRP 26 Quality Factors Versus RBE for Cell Death and Transformation

[One year in deep space at solar minimum for aluminum shielding]

x, g/cm^2	QF	RBE for cell death	RBE for cell transformation
0	7.1	7.1	6.4
1	5.6	7.0	6.4
2	5.3	6.9	6.3
3	5.1	6.8	6.3
5	4.7	6.7	6.2
10	3.9	6.5	6.0
20	3.2	6.2	5.7

Table 7. RBE for Cell Death and Transformation of C3H10T1/2 Cells for GCR Spectrum at Solar Minimum Behind Aluminum Shielding*

x, g/cm^2	RBE values for time periods of—		
	1 month	1 year	2 years
Cell death			
0	33.2 (37.0)	7.1	4.8 (4.6)
1	33.2 (36.1)	7.0	4.7 (4.5)
3	32.4 (35.1)	6.8	4.5 (4.3)
Cell transformation			
0	22.3 (22.2)	6.4	4.6 (4.5)
1	22.0 (22.2)	6.4	4.5 (4.5)
3	21.6 (21.8)	6.3	4.4 (4.4)

*Values in parentheses were scaled from 1-year RBE values using equation (18).

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15. Supplementary Notes

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16. Abstract

The assessment of biological damage from the galactic cosmic rays (GCR) is of current interest for exploratory-class space missions where the high-energy heavy ion (HZE) particles are the major concern. The relative biological effectiveness (RBE) determined by ground-based experiments with HZE particles is well described by a parametric track theory of cell inactivation. Using the track model and a deterministic GCR transport code, we consider the biological damage to mammalian cell cultures for 1 year in free space at solar minimum for typical spacecraft shielding. Included in this study are the effects of projectile and target fragmentation. The RBE values for the GCR spectrum that are fluence dependent in the track model are found to be more severe than currently used quality factors and are seen to obey a simple scaling law with the period of exposure in free space.

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